WHAT IS CLAIMED IS:

1. A method for identifying an agent that modulates the activity of a nuclear hormone receptor comprising the steps of:

providing a viable cell that expresses a nuclear hormone receptor and a nuclear hormone receptor substrate, reporter construct, or both, wherein expression of the substrate reporter construct is detectable and provides a measurement of nuclear hormone receptor pathway activity;

contacting a test cell with a test agent and an isolated bacterial product;

contacting a control cell with the isolated bacterial product in the absence of the agent;

detecting and comparing nuclear hormone receptor activity between the test and control cell to identify a test agent that interacts with the nuclear hormone receptor and modulates the activity of the nuclear hormone receptor by the bacterial product.

2. The method of claim 1, wherein the nuclear hormone receptor is selected from a glucocorticoid receptor (GR), androgen receptor (AR), mineralocorticoid receptor (MR), progestin receptor (PR), estrogen receptor (ER), thyroid hormone receptor (TR), vitamin D receptor (VDR), retinoid receptor (RAR or RXR), peroxisome receptor (XPAR or PPAR), icosanoid receptor (IRs), steroid receptor and thyroid receptor.

3. The method of claim 2, wherein the nuclear hormone receptor is GR.

4. The method of claim 2, wherein the nuclear hormone receptor is PR.

5. The method of claim 1, wherein the bacterial product is a bacterial wall protein, soluble bacterial protein, or lipopolysaccharide.

6. The method of claim 1, wherein the bacterial product is a bacterial toxin that is not endotoxin.

7. The method of claim 6, wherein the bacterial toxin elicits one or more symptoms of a toxic effect, inflammatory response, stress, shock, chronic sequelae, autoimmunity, or mortality in a susceptible host infected with a bacterium that produces the toxin.

8. The method of claim 6, wherein the bacterial toxin exhibits metalloprotease activity.

9. The method of claim 8, wherein the bacterial toxin is anthrax lethal factor (LF) or lethal toxin (LeTx) or a metalloenzyme of *Clostridium tetanus* or *C. botulinum* bacteria.

10. The method of claim 1, wherein the bacterial product is a bacterial antigen.

11. The method of claim 10, wherein the bacterial antigen is a pyrogenic toxin superantigen (PTSAg).

12. The method of claim 1, wherein the agent exerts its effect on the nuclear hormone receptor is through a mechanism other than inhibition of a MEK1 or MAPKK pathway.

13. The method of claim 1, wherein the agent is a genetically engineered or chemically modified variant or mimetic of the bacterial product, a drug, or a cofactor for the nuclear hormone receptor.

14. The method of claim 1, wherein the agent is effective following administration to a mammalian subject to reduce one or more inflammatory and/or autoimmune symptoms that can accompany exposure to the bacterial product or infection by a pathogen expressing the product.

15. The method of claim 1, wherein the isolated bacterial product alters the activity of the nuclear hormone receptor and does not alter number of nuclear hormone receptors on the viable cell.

16. A method for identifying an agent that inhibits nuclear hormone receptor repression by a bacterial product comprising the steps of:

providing viable cells that express a nuclear hormone receptor and a nuclear hormone substrate, a reporter construct, or both, wherein expression of the substrate, the reporter construct or both is detectable and provides a measurement of nuclear hormone receptor pathway activity;

contacting test cells cells with a test agent and a bacterial product;

contacting control cells with a bacterial product;

detecting and comparing nuclear hormone receptor pathway activity between the test and control cells to identify a test agent that inhibits repression of the receptor pathway activity by the bacterial product.

17. The method of claim 16, wherein the bacterial product is anthrax lethal factor (LF) or lethal toxin (LeTx).

18. The method of claim 17, wherein the agent that inhibits or blocks anthrax lethal factor (LF) or lethal toxin (LeTx) repression of nuclear hormone receptor activity is a cofactor for the nuclear hormone receptor.

19. The method of claim 18, wherein the cofactor is a coactivator for the nuclear hormone receptor.

20. The method of claim 18, wherein the nuclear hormone receptor is GR.

21. The method of claim 18, wherein the nuclear hormone receptor is PR.

22. The method of claim 18, wherein the nuclear hormone receptor is estrogen receptor-α (ER-α).

23. The method of claim 18, wherein the effective agent is a genetically engineered or chemically modified variant or mimetic of LF or LeTx, a drug, or a cofactor for the nuclear hormone receptor.

24. The method of claim 24, wherein the effective agent is a co-activator for the nuclear hormone receptor.

25. A method for identifying an active protein or other macromolecule from a cell expressesing a nuclear hormone receptor, wherein the active protein or other macromolecule interacts with a bacterial product that modulates nuclear hormone receptor pathway activity, comprising the steps of:

exposing the bacterial product to a lysate or other biological sample from the cell expressing the nuclear hormone receptor under conditions to allow for binding of the bacterial product to the active protein or other macromolecule;

contacting the bacterial product with a binding partner that provides for isolation or identification of the bacterial product bound to the active protein or other macromolecule;

detecting a bound complex of the bacterial product with the active protein or other macromolecule; and

identifying the active protein or other macromolecule bound in the complex.

26. The method of claim 25, wherein the binding partner is a polyclonal or monoclonal antibody that binds the bacterial product.

27. The method of claim 32 which comprises an immunoprecipitation assay.

28. The method of claim 25, wherein the active protein or other macromolecule bound in the complex is identified before separation from the complex, or following an additional step to separate the active protein or other macromolecule from the complex.

29. The method of claim 31, wherein the active protein or other macromolecule bound in the complex is identified by Western blotting and/or mass spectroscopy.

30. A method for alleviating or preventing one or more symptoms of a bacterial disease, inflammatory reaction, or autoimmune response in a mammalian subject comprising administering an effective amount of an agonist or antagonist of a nuclear hormone receptor selected according to the method of claim 1.

31. A method for alleviating or preventing one or more symptoms of a bacterial disease, inflammatory reaction, or autoimmune response in a mammalian subject comprising administering an effective amount of an agent that inhibits or enhances modulation of a nuclear hormone receptor by a bacterial product.

32. A method for alleviating or preventing one or more symptoms of anthrax disease and/or an associated inflammatory reaction, or autoimmune response, in a mammalian subject comprising administering an effective amount of an effective agent that inhibits, blocks, or enhances modulation of activity of one or more nuclear hormone receptor(s) by a anthrax lethal factor (LF) or lethal toxin (LeTx) or an analog, variant, derivative, or mimetic thereof.

33. A method for alleviating or preventing one or more symptoms of a bacterial disease, inflammatory reaction, or autoimmune response in a mammalian subject comprising administering an effective amount of a cofactor that is an agonist or antagonist of a nuclear hormone binding receptor.

34. A pharmaceutical composition for alleviating or preventing one or more symptoms of a bacterial disease, inflammatory reaction, or autoimmune response in a mammalian subject comprising an effective amount of an agonist or antagonist of a nuclear hormone receptor selected according to the method of claim 1.

35. A pharmaceutical composition for alleviating or preventing one or more symptoms of a bacterial disease, inflammatory reaction, or autoimmune response in a mammalian subject comprising an effective amount of an agent that inhibits or enhances modulation of a nuclear hormone receptor by a bacterial product.

36. The method of claim 35, wherein the agent is a cofactor of the nuclear hormone receptor.

37. A pharmaceutical composition for alleviating or preventing one or more symptoms of anthrax disease and/or an associated inflammatory reaction, or autoimmune response, in a mammalian subject comprising an effective amount of an effective agent that inhibits, blocks or

enhances modulation of activity of one or more nuclear hormone receptor(s) by an anthrax lethal factor (LF) or lethal toxin (LeTx) or an analog, variant, derivative, or mimetic thereof.

38. A composition comprising a recombinantly or chemically modified analog, fragment or derivative of a bacterial product that exhibits substantially reduced or increased activity as a modulator of nuclear hormone receptor activity compared to a native or wild-type counterpart bacterial product.

39. The composition of claim 38, wherein the composition elicits an immune response against the native or wild-type counterpart bacterial product in a mammalian subject

40. The immunogenic composition of claim 38, wherein said analog, fragment or derivative comprises a mutant variant, truncated fragment, or chemically modified derivative of an anthrax lethal factor (LF) or lethal toxin (LeTx).

41. The immunogenic composition of claim 40, wherein said LF or LeTx variant, fragment or derivative exhibits substantially reduced or increased activity for GR and/or PR repression.

42. The immunogenic composition of claim 40, wherein said LF or LeTx variant, fragment or derivative exhibits substantial activity as an immunogen, and/or inhibits, blocks, or enhances nuclear hormone repression activity by native LF or LeTx.

43. The immunogenic composition of claim 38, wherein said analog, fragment or derivative is characterized by a reduction or increase in a level of nuclear hormone modulation activity of at least 30% compared to repressor modulation activity of a corresponding native bacterial product.

44. A composition comprising a recombinantly or chemically modified analog, fragment or derivative of a bacterial product that inhibits, blocks, or enhances an interaction of a corresponding native bacterial product with a nuclear hormone receptor.

45. A method for identifying an agent of use in treating anthrax, comprising:

providing viable cells that express a receptor selected from the group consisting of a glucocorticoid receptor, an estrogen receptor α (ER-α), and a progresterone receptor B (PR-B) and a nucleic acid comprising a responsive element selected from the group consisting of a glucocorticoid receptor responsive element, an estrogen receptor α (ER-α) responsive element, and a progresterone receptor B (PR-B) responsive element, respectively, wherein the responsive element is operably linked to a nucleic acid encoding a polypeptide, wherein expression of the polypeptide is detectable and provides a measurement of the activity of the glucocorticoid responsive element;

contacting test cells with a test agent and anthrax lethal toxin (LeTx);

detecting expression of the polypeptide, wherein increased expression of the polypeptide as compared to a control identifies the agent as of use in treating anthrax.

46. The method of claim 45, wherein the control is a test cell contacted with anthrax lethal toxin in the absence of the agent.

47. The method of claim 45, wherein the receptor comprises a glucocoriticoid receptor and wherein the responsive element is a glucocorticoid responsive element.

48. The method of claim 45, wherein the receptor comprises a estrogen receptor α (ER-α) and the responsive element comprises a estrogen receptor α (ER-α) responsive element.

49. The method of claim 45, wherein the receptor is a progesterone receptor B (PR-b) and the responsive element comprises a progesterone receptor B responsive element.

50. A agonist of the glucocorticoid receptor, an estrogen receptor α (ER-α), and a progresterone receptor B (PR-B) in the manufacture of a medicament for the treatment of anthrax.

51. A method for treating an anthrax infection, comprising administering to a subject infected with anthrax or at risk of infection with anthrax a therapeutically effective amount of an agent that affects the activity of the glucocorticoid receptor, an estrogen receptor α (ER-α), and a progresterone receptor B (PR-B), thereby treating the anthrax infection.